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NEWS	12	MAY 30	DGENE, PCTGEN, and USGENE enhanced with new homology sequence search option
NEWS	13	JUN 06	EPFULL enhanced with 260,000 English abstracts
NEWS	14	JUN 06	KOREAPAT updated with 41,000 documents
NEWS	15	JUN 13	USPATFULL and USPAT2 updated with 11-character patent numbers for U.S. applications
NEWS	16	JUN 19	CAS REGISTRY includes selected substances from web-based collections
NEWS	17	JUN 25	CA/CAPLUS and USPAT databases updated with IPC reclassification data
NEWS	18	JUN 30	AEROSPACE enhanced with more than 1 million U.S. patent records
NEWS	19	JUN 30	EMBASE, EMBAL, and LEMBASE updated with additional options to display authors and affiliated organizations
NEWS	20	JUN 30	STN on the Web enhanced with new STN AnaVist Assistant and BLAST plug-in
NEWS	21	JUN 30	STN AnaVist enhanced with database content from EPFULL
NEWS	22	JUL 28	CA/CAPLUS patent coverage enhanced
NEWS	23	JUL 28	EPFULL enhanced with additional legal status information from the EPOline Register
NEWS	24	JUL 28	IFICDB, IFIPAT, and IFIUDB reloaded with enhancements
NEWS	25	JUL 28	STN Viewer performance improved
NEWS	26	AUG 01	INPADOCDB and INPAFAMDB coverage enhanced
NEWS	27	AUG 13	CA/CAPLUS enhanced with printed Chemical Abstracts page images from 1967-1998
NEWS	28	AUG 15	CAOLD to be discontinued on December 31, 2008
NEWS	29	AUG 15	CAPLUS currency for Korean patents enhanced

NEWS EXPRESS JUNE 27 08 CURRENT WINDOWS VERSION IS V8.3,  
AND CURRENT DISCOVER FILE IS DATED 23 JUNE 2008.

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FILE 'HOME' ENTERED AT 17:22:19 ON 18 AUG 2008

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	ENTRY	SESSION
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FILE 'MEDLINE' ENTERED AT 17:22:30 ON 18 AUG 2008

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=> S (EVEC or fibulin-5 or DANCE) (8A) (integrin (4A) binding or bind or bound)  
L1 20 (EVEC OR FIBULIN-5 OR DANCE) (8A) (INTEGRIN (4A) BINDING OR BIND  
OR BOUND)

=> S (EVEC or fibulin-5 or DANCE) (6A) (domain or motif or fragment or region or  
section or portion or segment or part)  
L2 96 (EVEC OR FIBULIN-5 OR DANCE) (6A) (DOMAIN OR MOTIF OR FRAGMENT  
OR REGION OR SECTION OR PORTION OR SEGMENT OR PART)

=> L2 (P) (integrin (4A) binding or bind or bound)  
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L3 9 L2 (P) (INTEGRIN (4A) BINDING OR BIND OR BOUND)

=> duplicate  
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove  
ENTER L# LIST OR (END):13  
DUPLICATE PREFERENCE IS 'MEDLINE, EMBASE, BIOSIS, CAPLUS'  
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n

PROCESSING COMPLETED FOR L3  
L4 3 DUPLICATE REMOVE L3 (6 DUPLICATES REMOVED)

=> d 14 1-3 bib ab

L4 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN  
AN 2005:1075903 CAPLUS  
DN 143:344598  
TI Cleavage of fibulin-5/DANCE by a serine protease is essential for elastogenesis in vivo: DANCE cleavage assay and drug screening  
IN Nakamura, Tomoyuki; Hirai, Maretoshi  
PA Kyoto University, Japan  
SO PCT Int. Appl., 119 pp.  
CODEN: PIXXD2  
DT Patent  
LA Japanese  
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005093057	A1	20051006	WO 2005-JP4274	20050304
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2561495	A1	20051006	CA 2005-2561495	20050304
	EP 1762610	A1	20070314	EP 2005-720545	20050304
	R: CH, DE, FR, GB, IT, LI				
	CN 1961073	A	20070509	CN 2005-80017559	20050304
	US 20070218003	A1	20070920	US 2006-594339	20060927
PRAI	JP 2004-96685	A	20040329		
	WO 2005-JP4274	W	20050304		
AB	A method of screening that enables development of a medicine having a novel mechanism of action capable of regulating generation of an elastic fiber tissue; and various means that are requisite for the method; are provided. In particular, there are provided a polypeptide obtained by cleaving DANCE (developmental arteries and neural crest epidermal growth factor (EGF)-like) and a polynucleotide coding for the polypeptide; a method of cleaving DANCE; an antibody against the polypeptide obtained by cleaving DANCE; a method of measuring the amount of DANCE cleavage and a kit therefor; a DANCE variant and polynucleotide coding for the same; various DANCE complexes and a method of preparing the same; a method of screening a substance capable of regulating the activity of DANCE or a DANCE-specific protease, and a substance obtained by the screening method; an agent for regulating generation of an elastic fiber tissue; a kit comprising at least DANCE and a polynucleotide coding for the same; etc. Examples, etc. describe that DANCE is in vitro or in vivo cleaved by a serine protease, and that with respect to truncated DANCE, DANCE bind to each other and to LTBP2 (latent TGF- $\beta$ -binding protein 2) and lysyl oxidase (LOX). It is presumed that (1) it no longer binds to cell surface integrin, (2) DANCE mols. no longer bind to each other and (3) the binding to LTBP2 is stronger than that to full-length DANCE (see Consideration 3), and that DANCE having an amino terminal domain cleaved exhibits substantially no activity of generating of elastic fibrous tissue (see Referential Example 2). Consequently, it is				

proved that the amino terminal domain of DANCE is needed for generating of elastic fibrous tissue.

RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 3 MEDLINE on STN DUPLICATE 1  
AN 2004591099 MEDLINE  
DN PubMed ID: 15528465  
TI Fibulin-5 is a novel binding protein for extracellular superoxide dismutase.  
AU Nguyen Andrew D; Itoh Shinichi; Jeney Viktoria; Yanagisawa Hiromi; Fujimoto Mitsuo; Ushio-Fukai Masuko; Fukai Tohru  
CS Division of Cardiology, Department of Medicine, Emory University School of Medicine, Atlanta, Ga 30322, USA.  
NC HL58000 (United States NHLBI)  
R01 HL70187 (United States NHLBI)  
SO Circulation research, (2004 Nov 26) Vol. 95, No. 11, pp. 1067-74.  
Electronic Publication: 2004-11-04.  
Journal code: 0047103. E-ISSN: 1524-4571.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
LA English  
FS Priority Journals  
EM 200506  
ED Entered STN: 30 Nov 2004  
Last Updated on STN: 16 Jun 2005  
Entered Medline: 15 Jun 2005  
AB The extracellular superoxide dismutase (ecSOD) plays an important role in atherosclerosis and endothelial function by modulating levels of the superoxide anion ( $O_2^{\cdot-}$ ) in the extracellular space. Although heparan sulfate proteoglycan is an important ligand for ecSOD, little is known about other biological binding partners of ecSOD. The goal of this study was to identify novel proteins that interact with ecSOD. A yeast two-hybrid screening of a human aorta cDNA library using ecSOD as bait identified fibulin-5 as a predominant binding protein for ecSOD. Further analysis showed that the binding domain of ecSOD within fibulin-5 mapped to its C-terminal domain. In vitro pull-down assays and coimmunoprecipitation analysis further confirmed that ecSOD interacts with fibulin-5 in vitro and in vivo. Studies using fibulin-5 $^{-/-}$  mice indicated that fibulin-5 is required for binding of ecSOD to vascular tissue. Importantly, the decrease in tissue-bound ecSOD levels in aortas from fibulin-5 $^{-/-}$  mice was associated with an increase in vascular  $O_2^{\cdot-}$  levels. Furthermore, immunohistochemical analysis using ApoE $^{-/-}$  mice suggested a codistribution of ecSOD and fibulin-5 in atherosclerotic vessels. In summary, we provide in this study the first evidence that the ecSOD-fibulin-5 interaction is required for ecSOD binding to vascular tissues, thereby regulating vascular  $O_2^{\cdot-}$  levels. This interaction may represent a novel mechanism for controlling vascular redox state in the extracellular space in various cardiovascular diseases such as atherosclerosis and hypertension in which oxidative stress is increased.

L4 ANSWER 3 OF 3 MEDLINE on STN DUPLICATE 2  
AN 2002078083 MEDLINE  
DN PubMed ID: 11805834  
TI Fibulin-5 is an elastin-binding protein essential for elastic fibre development in vivo.  
AU Yanagisawa Hiromi; Davis Elaine C; Starcher Barry C; Ouchi Takashi;

Yanagisawa Masashi; Richardson James A; Olson Eric N  
 CS Department of Molecular Biology, University of Texas Southwestern Medical  
 Center at Dallas, Texas 75390, USA.. hyanagisawa@aol.com  
 SO Nature, (2002 Jan 10) Vol. 415, No. 6868, pp. 168-71.  
 Journal code: 0410462. ISSN: 0028-0836.  
 CY England: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
 LA English  
 FS Priority Journals  
 EM 200202  
 ED Entered STN: 28 Jan 2002  
 Last Updated on STN: 15 Feb 2002  
 Entered Medline: 14 Feb 2002  
 AB Extracellular elastic fibres provide mechanical elasticity to tissues and  
 contribute towards the processes of organ remodelling by affecting  
 cell-cell signalling. The formation of elastic fibres requires the  
 assembly and crosslinking of tropoelastin monomers, and organization of  
 the resulting insoluble elastin matrix into functional fibres. The  
 molecules and mechanisms involved in this process are unknown. Fibulin-5  
 (also known as EVEC/DANCE) is an extracellular matrix protein abundantly  
 expressed in great vessels and cardiac valves during embryogenesis, and in  
 many adult tissues including the aorta, lung, uterus and skin, all of  
 which contain abundant elastic fibres. Here we show that fibulin-5 is a  
 calcium-dependent, elastin-binding protein that localizes to the surface  
 of elastic fibres in vivo. fibulin-5-/- mice develop marked elastinopathy  
 owing to the disorganization of elastic fibres, with resulting loose skin,  
 vascular abnormalities and emphysematous lung. This phenotype, which  
 resembles the cutis laxa syndrome in humans, reveals a critical function  
 for fibulin-5 as a scaffold protein that organizes and links elastic  
 fibres to cells. This function may be mediated by the RGD motif  
 in fibulin-5, which binds to cell surface integrins,  
 and the Ca2+-binding epidermal growth factor (EGF) repeats, which  
 bind elastin.

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PROCESSING COMPLETED FOR L2
L5          66 DUPLICATE REMOVE L2 (30 DUPLICATES REMOVED)
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DUPLICATE PREFERENCE IS 'MEDLINE, EMBASE, BIOSIS, CAPLUS'
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PROCESSING COMPLETED FOR L1
L6          7 DUPLICATE REMOVE L1 (13 DUPLICATES REMOVED)
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=> d l6 1-7 bib ab
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L6  ANSWER 1 OF 7      MEDLINE on STN          DUPLICATE 1
AN  2007035536      MEDLINE
DN  PubMed ID: 17130242
TI  Molecular analysis of fibulin-5 function during de novo synthesis of
    elastic fibers.
AU  Zheng Qian; Davis Elaine C; Richardson James A; Starcher Barry C; Li
```

Tiansen; Gerard Robert D; Yanagisawa Hiromi  
 CS Department of Molecular Biology, University of Texas Southwestern Medical  
 Center, 5323 Harry Hines Blvd., Dallas, TX 75390-9148, USA.  
 NC HL 071157 (United States NHLBI)  
 SO Molecular and cellular biology, (2007 Feb) Vol. 27, No. 3, pp. 1083-95.  
 Electronic Publication: 2006-11-27.  
 Journal code: 8109087. ISSN: 0270-7306.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)  
 (RESEARCH SUPPORT, NON-U.S. GOV'T)  
 LA English  
 FS Priority Journals  
 EM 200703  
 ED Entered STN: 20 Jan 2007  
 Last Updated on STN: 6 Mar 2007  
 Entered Medline: 5 Mar 2007  
 AB Elastic fibers contribute to the structural support of tissues and to the  
 regulation of cellular behavior. Mice deficient for the fibulin-5 gene  
 (fbln5(-/-)) were used to further elucidate the molecular mechanism of  
 elastic fiber assembly. Major elastic fiber components were present in  
 the skin of fbln5(-/-) mice despite a dramatic reduction of mature elastic  
 fibers. We found that fibulin-5 preferentially  
 bound the monomeric form of elastin through N-terminal and  
 C-terminal elastin-binding regions and to a preexisting matrix scaffold  
 through calcium-binding epidermal growth factor (EGF)-like (CB-EGF)  
 domains. We further showed that adenovirus-mediated gene transfer of  
 fbln5 was sufficient to regenerate elastic fibers and increase elastic  
 fiber-cell connections in vivo. A mutant fibulin-5 lacking the first 28  
 amino acids of the first CB-EGF domain, however, was unable to rescue  
 elastic fiber defects. Fibulin-5 thus serves as an adaptor molecule  
 between monomeric elastin and the matrix scaffold to aid in elastic fiber  
 assembly. These results also support the potential use of fibulin-5 as a  
 therapeutic agent for the treatment of elastinopathies.

L6 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN  
 AN 2006:223863 CAPLUS  
 TI Construction of Electrocatalytic Electrodes Bearing the Triphenylamine  
 Nucleus Covalently Bound to Carbon. A Halogen Dance in  
 Protonated Aminotriphenylamines  
 AU Mayers, Brian T.; Fry, Albert J.  
 SO Organic Letters (2006), 8(6), 1253  
 CODEN: ORLEF7; ISSN: 1523-7060  
 PB American Chemical Society  
 DT Journal; Errata  
 LA English  
 AB Unavailable

L6 ANSWER 3 OF 7 MEDLINE on STN DUPLICATE 2  
 AN 2006051293 MEDLINE  
 DN PubMed ID: 16435847  
 TI Construction of electrocatalytic electrodes bearing the triphenylamine  
 nucleus covalently bound to carbon. A halogen dance in  
 protonated aminotriphenylamines.  
 AU Mayers Brian T; Fry Albert J  
 CS Chemistry Department, Wesleyan University, Middletown, Connecticut 06459,  
 USA.  
 SO Organic letters, (2006 Feb 2) Vol. 8, No. 3, pp. 411-4.  
 Journal code: 100890393. ISSN: 1523-7060.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)

LA English  
 FS NONMEDLINE; PUBMED-NOT-MEDLINE  
 EM 200610  
 ED Entered STN: 27 Jan 2006  
 Last Updated on STN: 19 Oct 2006  
 Entered Medline: 18 Oct 2006

AB [reaction: see text]. The triarylamine nucleus has been attached to a carbon fiber electrode by diazotization of an aminotriphenylamine followed by electrochemical reduction. The resulting electrodes can electrocatalyze the oxidation of organic substrates. In acid, 4-amino-4',4''-dibromotriphenylamine undergoes dismutation into a mixture of amines containing from 0 to 3 bromine atoms.

L6 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN  
 AN 2005:1075903 CAPLUS

DN 143:344598

TI Cleavage of fibulin-5/DANCE by a serine protease is essential for elastogenesis in vivo: DANCE cleavage assay and drug screening

IN Nakamura, Tomoyuki; Hirai, Maretoshi

PA Kyoto University, Japan

SO PCT Int. Appl., 119 pp.

CODEN: PIXXD2

DT Patent

LA Japanese

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005093057	A1	20051006	WO 2005-JP4274	20050304
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2561495	A1	20051006	CA 2005-2561495	20050304
	EP 1762610	A1	20070314	EP 2005-720545	20050304
	R: CH, DE, FR, GB, IT, LI				
	CN 1961073	A	20070509	CN 2005-80017559	20050304
	US 20070218003	A1	20070920	US 2006-594339	20060927
PRAI	JP 2004-96685	A	20040329		
	WO 2005-JP4274	W	20050304		

AB A method of screening that enables development of a medicine having a novel mechanism of action capable of regulating generation of an elastic fiber tissue; and various means that are requisite for the method; are provided. In particular, there are provided a polypeptide obtained by cleaving DANCE (developmental arteries and neural crest epidermal growth factor (EGF)-like) and a polynucleotide coding for the polypeptide; a method of cleaving DANCE; an antibody against the polypeptide obtained by cleaving DANCE; a method of measuring the amount of DANCE cleavage and a kit therefor; a DANCE variant and polynucleotide coding for the same; various DANCE complexes and a method of preparing the same; a method of screening a substance capable of regulating the activity of DANCE or a DANCE-specific protease, and a substance obtained by the screening method; an agent for regulating generation of an elastic fiber tissue; a kit comprising at least DANCE and a polynucleotide coding for the same; etc. Examples, etc. describe that DANCE is in vitro or in vivo cleaved by a serine protease,

and that with respect to truncated DANCE, DANCE bind to each other and to LTBP2 (latent TGF- $\beta$ -binding protein 2) and lysyl oxidase (LOX). It is presumed that (1) it no longer binds to cell surface integrin, (2) DANCE mols. no longer bind to each other and (3) the binding to LTBP2 is stronger than that to full-length DANCE (see Consideration 3), and that DANCE having an amino terminal domain cleaved exhibits substantially no activity of generating of elastic fibrous tissue (see Referential Example 2). Consequently, it is proved that the amino terminal domain of DANCE is needed for generating of elastic fibrous tissue.

RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 5 OF 7 MEDLINE on STN DUPLICATE 3  
AN 2004591099 MEDLINE  
DN PubMed ID: 15528465  
TI Fibulin-5 is a novel binding protein for extracellular superoxide dismutase.  
AU Nguyen Andrew D; Itoh Shinichi; Jeney Viktoria; Yanagisawa Hiromi; Fujimoto Mitsunaki; Ushio-Fukai Masuko; Fukai Tooru  
CS Division of Cardiology, Department of Medicine, Emory University School of Medicine, Atlanta, Ga 30322, USA.  
NC HL58000 (United States NHLBI)  
R01 HL70187 (United States NHLBI)  
SO Circulation research, (2004 Nov 26) Vol. 95, No. 11, pp. 1067-74.  
Electronic Publication: 2004-11-04.  
Journal code: 0047103. E-ISSN: 1524-4571.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
LA English  
FS Priority Journals  
EM 200506  
ED Entered STN: 30 Nov 2004  
Last Updated on STN: 16 Jun 2005  
Entered Medline: 15 Jun 2005  
AB The extracellular superoxide dismutase (ecSOD) plays an important role in atherosclerosis and endothelial function by modulating levels of the superoxide anion (O<sub>2</sub><sup>-</sup>) in the extracellular space. Although heparan sulfate proteoglycan is an important ligand for ecSOD, little is known about other biological binding partners of ecSOD. The goal of this study was to identify novel proteins that interact with ecSOD. A yeast two-hybrid screening of a human aorta cDNA library using ecSOD as bait identified fibulin-5 as a predominant binding protein for ecSOD. Further analysis showed that the binding domain of ecSOD within fibulin-5 mapped to its C-terminal domain. In vitro pull-down assays and coimmunoprecipitation analysis further confirmed that ecSOD interacts with fibulin-5 in vitro and in vivo. Studies using fibulin-5<sup>-/-</sup> mice indicated that fibulin-5 is required for binding of ecSOD to vascular tissue. Importantly, the decrease in tissue-bound ecSOD levels in aortas from fibulin-5<sup>-/-</sup> mice was associated with an increase in vascular O<sub>2</sub><sup>-</sup> levels. Furthermore, immunohistochemical analysis using ApoE<sup>-/-</sup> mice suggested a codistribution of ecSOD and fibulin-5 in atherosclerotic vessels. In summary, we provide in this study the first evidence that the ecSOD-fibulin-5 interaction is required for ecSOD binding to vascular tissues, thereby regulating vascular O<sub>2</sub><sup>-</sup> levels. This interaction may represent a novel mechanism for controlling vascular redox state in the extracellular space in various cardiovascular diseases such as atherosclerosis and hypertension in which oxidative stress is



increased.

L6 ANSWER 6 OF 7 MEDLINE on STN DUPLICATE 4  
AN 2004329556 MEDLINE  
DN PubMed ID: 15231070  
TI Fibulin-5 antagonizes vascular endothelial growth factor (VEGF) signaling and angiogenic sprouting by endothelial cells.  
AU Albig Allan R; Schiemann William P  
CS Program in Cell Biology, Department of Pediatrics, National Jewish Medical and Research Center, Denver, Colorado, USA.  
NC CA095519 (United States NCI)  
CA99321 (United States NCI)  
SO DNA and cell biology, (2004 Jun) Vol. 23, No. 6, pp. 367-79.  
Journal code: 9004522. ISSN: 1044-5498.  
CY United States  
DT (COMPARATIVE STUDY)  
Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, NON-U.S. GOV'T)  
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
LA English  
FS Priority Journals  
EM 200408  
ED Entered STN: 3 Jul 2004  
Last Updated on STN: 4 Aug 2004  
Entered Medline: 3 Aug 2004  
AB Fibulin-5 (FBLN-5) is a widely expressed, integrin-binding extracellular matrix protein that mediates endothelial cell adhesion and scaffolds cells to elastic fibers. It is also a gene target of TGF-beta in fibroblasts and endothelial cells that regulates cell proliferation and motility in a context-specific manner. Whereas FBLN-5 expression is low in adult vasculature, its expression is high in developing and injured vasculature, implicating FBLN-5 in regulating angiogenesis and endothelial cell function. We show here that TGF-beta stimulates FBLN-5 expression in endothelial cells, and that this response was inhibited by coadministration of the proangiogenic factor, VEGF. FBLN-5 expression was downregulated significantly during endothelial cell tubulogenesis, implying that FBLN-5 expression antagonizes angiogenesis. Accordingly, FBLN-5 overexpression in or recombinant FBLN-5 treatment of endothelial cells abrogated their ability to undergo angiogenic sprouting, doing so by inhibiting endothelial cell proliferation and invasion through Matrigel matrices. Moreover, FBLN-5 antagonized VEGF signaling in endothelial cells, as well as enhanced their expression of the antiangiogenic factor, thrombospondin-1. Finally, the ability of FBLN-5 to antagonize angiogenic processes was determined to be independent of its integrin-binding RGD motif. Collectively, our findings establish FBLN-5 as a novel antagonist of angiogenesis and endothelial cell activities, and offer new insights into why tumorigenesis downregulates FBLN-5 expression.

L6 ANSWER 7 OF 7 MEDLINE on STN DUPLICATE 5  
AN 2002400945 MEDLINE  
DN PubMed ID: 12021267  
TI Context-specific effects of fibulin-5 (DANCE/EVEC) on cell proliferation, motility, and invasion. Fibulin-5 is induced by transforming growth factor-beta and affects protein kinase cascades.  
AU Schiemann William P; Blobel Gerard C; Kalume Dario E; Pandey Akhilesh; Lodish Harvey F  
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AB FIBLIN-5 (FBLN-5; also known as DANCE or EVEC) is an integrin-binding extracellular matrix protein that mediates endothelial cell adhesion; it is also a calcium-dependent elastin-binding protein that scaffolds cells to elastic fibers, thereby preventing elastinopathy in the skin, lung, and vasculature. Transforming growth factor-beta (TGF-beta) regulates the production of cytokines, growth factors, and extracellular matrix proteins by a variety of cell types and tissues. We show here that TGF-beta stimulates murine 3T3-L1 fibroblasts to synthesize FBLN-5 transcript and protein through a Smad3-independent pathway. Overexpression of FBLN-5 in 3T3-L1 cells increased DNA synthesis and enhanced basal and TGF-beta-stimulated activation of ERK1/ERK2 and p38 mitogen-activated protein kinase (MAPK). FBLN-5 overexpression also augmented the tumorigenicity of human HT1080 fibrosarcoma cells by increasing their DNA synthesis, migration toward fibronectin, and invasion through synthetic basement membranes. In stark contrast, FBLN-5 expression was down-regulated in the majority of metastatic human malignancies, particularly in cancers of the kidney, breast, ovary, and colon. Unlike its proliferative response in fibroblasts, FBLN-5 overexpression in mink lung Mv1Lu epithelial cells resulted in an antiproliferative response, reducing their DNA synthesis and cyclin A expression. Moreover, FBLN-5 synergizes with TGF-beta in stimulating AP-1 activity in Mv1Lu cells, an effect that was abrogated by overexpression of dominant-negative versions of either MKK1 or p38 MAPKalpha. Accordingly, both the stimulation and duration of ERK1/ERK2 and p38 MAPK by TGF-beta was enhanced in Mv1Lu cells expressing FBLN-5. Our findings identify FBLN-5 as a novel TGF-beta-inducible target gene that regulates cell growth and motility in a context-specific manner and affects protein kinase activation by TGF-beta. Our findings also indicate that aberrant FBLN-5 expression likely contributes to tumor development in humans.